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Syphilis as AIDS?—A Call for Research

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Abstract—Based upon a literature survey, this paper outlines the argument that *Treponema pallidum* may be the etiologic agent of AIDS. AIDS and syphilis have similar epidemiology, immunology and pathologies. In addition, the current methods of detecting and treating syphilis are inadequate. The similarities between syphilis and AIDS support the need for further research into their relationships, including the role *Treponema pallidum* may play in AIDS etiology.

Introduction

Most people currently believe that acquired immunodeficiency syndrome (AIDS) is caused by human immunodeficiency virus (HIV) However, as outlined by several authors (1–3), there is medical data to support the conclusion that *Treponema pallidum* (TP), the etiologic agent of syphilis, may be the cause of AIDS.

Discussion

1. Microbial etiology is determined according to Koch's postulates: 1) the microbe in question must be regularly detected in patients with the disease, 2) the microbe can be isolated in pure cultures from patients 3) the pure cultures when inoculated into susceptible animals will produce the disease, and 4) the microbe can again be isolated in pure cultures from the inoculated animals. These postulates have never been fulfilled for HIV-induced AIDS (4–7). Dr Robert Gallo's 1984 assertion that HIV is the primary cause of AIDS was only based upon a 40% incidence of HIV in patients with AIDS (though a higher correlation rate was found in asymptomatic patients) (8). By contrast, TP was found in 73% of the AIDS patients in a study by researchers at the University of Miami (Florida) (9). Though HIV's strong correlation with AIDS has made it a part of the Centers for Disease Control (CDC) syndrome definition, if the virus does not cause AIDS, then aiming treatments at HIV will not cure AIDS.

2. AIDS and TP are both transmitted via blood products (transfusion, sharing needles), congenital infection (transplacental), and sexual practices (10). This epidemiologic similarity is not conclusive, as many other diseases share these common modes of transmission with AIDS. However, syphilis also has striking immunologic and pathologic similarities with AIDS.

3. Immunosuppression, a hallmark of AIDS, is also recognized as being characteristic of syphilis (11–13). One study has shown a 30% reduction of T-lymphocyte populations in syphilitic patients (14). During the sexual and drug revolutions of the 1960s and 1970s, sexually promiscuous homosexuals commonly suffered multiple, and often concurrent, infections of TP. Furthermore, syphilis was known to be epidemic in both the United States homosexual and Africal het-

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erosexual populations at the onset of AIDS era (15). Taken together, these factors could have exacerbated the 30% immunodepletion from TP infection into a pernicious state of complete immunosuppression.

4. The pathologies of AIDS and syphilis are strikingly similar, with virtually any organ a candidate for infection (10). Patients with either disease may manifest alopecia, anorexia, aphasia, ataxia, bladder disturbances, cranial nerve involvement including facial paralysis, dementia, encepahlitis, epilepsy, fever, glomerulonephritis, hemiplegia, hyperactive reflexes, Kaposi's sarcoma, laryngitis, lymphadenopathy, nephrotic syndrome, optic atrophy, paraplegia, pharyngitis, *Pneumocystis carinii pneumonia*, seizures, strokes, and thrush (16–24). In addition, it is now acknowledged that neurosyphilis may occur in any stage of TP infection (25).

5. Though quick and inexpensive (26), the standard blood tests used to diagnose syphilis are not reliable. The most common diagnostic tools used, the Venereal Disease Research Laboratory (VDRL) and Rapid Plasma Reagin (RPR), do not detect the actual presence of the treponeme itself. They assess immune system reactions commonly associated with TP infection (27). In syphilis, as in AIDS, the immune system is compromised and these tests produce a significant number of false negatives (10, 27–29).

6. Syphilis cannot be cured with penicillin G. First, penicillin G is unable to go everywhere that TP goes in the body. TP invades three distinct compartments: the blood and lymph circulatory systems soon after infection (10), the intracellular environment of various cells (30, 31) within 30 min of infection (32), and the central nervous system as early as 2 weeks from infection (10). While penicillin G can effectively eradicate the circulatory infections, this antibiotic does not readily enter cells (33, 34), and benzathine penicillin G, the most commonly used penicillin, cannot cross the blood-brain-barrier to establish treponemicidal levels in the central nervous system (35–38).

Second, penicillin cannot cure syphilis because it only kills TP when the bacterium is actively dividing. The current CDC protocols for treating syphilis are based upon a doubling time of 30–33 h (39, 40). It has been suggested that the dividing time of TP may be elongated to months in advanced syphilis (41, 42), leaving virulent treponema present in vivo long after antibiotic serum levels have dissipated.

Finally, evidence of recurring syphilis in patients treated with penicillin G supports the conclusion that this drug does not eradicate TP infection (43–51). Research into other known treponemacidal compounds (52) such as doxycycline, which better permeates the blood-brain-barrier than benzathine penicillin G (53), and erythromycin, which easily permeates cell membranes (33, 34), might yield syphilis treatment protocols of longer duration and greater efficacy.

Conclusion

Given that AIDS and syphilis have similar epidemiology, immunology and pathology, the question remains: can *Treponema pallidum* cause AIDS? The literature suggests that syphilis, which is often undetected or ineffectively treated when diagnosed, may become clinically indistinguishable from AIDS (54, 55). Surely, the data presented warrants further research.

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